Allergic Dermatitis

See Articles:
Hnilica KA, Angarano DW. Advances in Immunology: Role of T-helper Lymphocyte Subsets. Compendium of Continuing Education for the Practicing Veterinarian. 19:1;87-93, 1997.
Hnilica, K. A. Advances in allergy diagnosis and treatment. The Compendium. 1998. 20: 258-259

I. Allergy Immunology

- hypersensitivity is an exaggerated immunologic response to a foreign antigen

A. Classic hypersensitivity reactions

1. Type I (anaphylactic, immediate)
   IgE Antibodies fixed to mast cells react with antigen, triggering the release of histamine and other mediators (i.e. urticaria, atopy)

2. Type II (cytotoxic)
   IgG or IgM antibodies react with antigen on target cells and activate complement causing cell lysis (i.e. AIHA, ITP)

3. Type III (immune complex)
   IgG or IgM antibodies form complexes with antigen and complement generating neutrophil chemotactic factors with resultant tissue inflammation (i.e. SLE, Rheumatoid arthritis)

4. Type IV (cell-mediated, delayed)
   sensitized T lymphocytes react with antigen producing inflammation through action of lymphokine (i.e. allergic contact dermatitis)
B. Current concept

Depending on the antigen, route of exposure, antigen presenting cells, and tissue type, different immunological responses can be triggered. T-lymphocytes (helper cells) initiate and drive the immune response in reaction to the foreign antigens identified in the tissues (skin, nasal mucosa, lungs).

1. **Langerhan’s cells** – dendritic cells that survey the epidermis for foreign antigens which are then processed and presented to the Th2 cells

2. **T helper lymphocytes subset 2 (Th2 cells)** – T cell responsible for driving the allergic mechanism through cytokine production (Interleukin-4, IL-10). Triggers B cells to produce IgE, stimulates mast cells and eosinophils, activates Langerhan’s cells, and promotes additional Th2 cells activation.

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**T - helper Lymphocytes**

- **Th1 cells**
  - infections
  - autoimmunity
  - multiple sclerosis
  - rheumatoid arthritis
  - psoriasis

- **Th2 cells**
  - parasitic infections
  - allergy
  - SLE
  - shingles
  - AIDS
3. **B lymphocytes** – activated to produce IgE

4. **Mast cells** – binds IgE on its surface to screen for antigens in the skin. When two IgE molecules are cross linked, the mast cell will degranulate, releasing its inflammatory mediators (histamine, heparin, proteases, prostaglandins, leukotrienes, and many, many others).

5. **Eosinophils** – proinflammatory cell for the allergic reaction

6. **Keratinocytes** – when damaged by excoriation, the keratinocytes release many inflammatory mediators (prostaglandins, leukotrienes, mast cell activating factor, etc.) that promote the allergic response.

- **Itch**
  - **Epidermal damage**
    - prostaglandin
    - leukotrienes
    - IL-4
  - **Nerve stimulation**
    - substance P